



## Case Report

## Transient global amnesia complicating pulmonary function testing

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## ABSTRACT

Transient global amnesia (TGA), a disorder characterized by the acute onset of anterograde and retrograde amnesia, is well known to neurologists and has been reported in association with a wide range of triggers. We report a patient who experienced the onset of TGA during pulmonary function testing. Potential mechanisms and other scenarios provoking TGA relevant to the pulmonologist, such as bronchoscopy, exercise testing, high altitude, and the use of phosphodiesterase inhibitors are discussed.

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## 1. Introduction

Transient global amnesia (TGA) is an uncommon neurological disorder, consisting of the acute onset of anterograde and retrograde amnesia that resolves within 24 h.<sup>1</sup> There is increasing recognition of potential triggers for TGA, including circumstances relevant to pulmonologists, which may help to elucidate its underlying pathophysiology. Here we present a patient who developed TGA during pulmonary function testing (PFT).

## 2. Case report

A 60 year old woman with a past medical history of hypertension, depression, and migraine without aura consulted with a pulmonologist for a 1 year history of unexplained cough. She was not a smoker and had no history of asthma or gastroesophageal reflux. After a normal physical examination and chest radiography, she was referred for PFT. Immediately after the first few breaths of spirometry, she became acutely confused. She appeared calm, but stopped breathing into the mask, asking questions repeatedly such as, “Where am I?” “What is going on?” She denied headache and dyspnea, and was immediately taken to the emergency room.

Upon arrival, she was afebrile, had normal vital signs, and an unremarkable physical examination. She was lucid, recognized her husband, followed complex requests, and recalled all of the

prior evening’s activities. Despite repeated explanations of her current circumstances, she continued to question where she was and what was happening. Her neurological examination was otherwise normal. Computed tomography of the brain, electrocardiogram, serum chemistry, complete blood count, liver function and thyroid function tests were all normal. Brain magnetic resonance imaging (MRI) was deferred because of claustrophobia. Over several hours the patient gradually returned to normal, although she remained permanently amnesic for an 8 h block of time that day. Five years have elapsed since experiencing TGA and the patient has had no recurrence.

## 3. Discussion

This patient experienced the acute onset of a transient disturbance of strictly episodic memory, which was typical of TGA. The notable feature in this patient was the onset of symptoms during PFT. Precipitating events, most commonly emotional stress, physical exertion, and abrupt temperature change, may occur in 26–65% of patients presenting with TGA.<sup>1</sup>

The annual incidence of TGA is 5–11 per 100,000 persons.<sup>1</sup> The pathogenesis of TGA is unknown, with putative etiologies related to cerebral arterial ischemia, venous congestion, seizures, migraine, and psychological stressors. 11–84% of TGA patients have MRI diffusion-weighted imaging lesions (possibly signaling ischemia) in the mesiotemporal lobe, which localizes well to the hippocampus, the site of episodic memory formation and consolidation. However, even in individuals with these MRI lesions, arterial ischemia is doubtful because they do not have an increased vascular risk profile and their MRI show no evidence of other ischemic brain changes.<sup>2</sup>

Abbreviations: MRI, magnetic resonance imaging; PFT, pulmonary function testing; TGA, transient global amnesia.

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The most intriguing etiology for TGA is related to venous congestion. Internal jugular venous insufficiency may allow for increased venous pressure to be transmitted during Valsalva maneuver, causing ischemia to the hippocampus by venous congestion. Patients with TGA are more likely than controls to have jugular venous insufficiency or reflux during Valsalva when measured by duplex ultrasonography, with retrograde flow patterns.<sup>1</sup> Our patient may have had vulnerability because of underlying venous insufficiency. After forced exhalations during PFT, an acute escalation of intrathoracic pressure likely occurred, causing jugular and intracranial venous congestion, and hippocampal ischemia. Alternatively, the stress of a medical test was a trigger for TGA. A migrainous etiology is doubtful as the patient experienced no headache, never had aura, and had no long-term recurrence.

TGA occurring in PFT has been reported in 1 other patient, but the specific details were not published.<sup>3</sup> Other triggers relevant to the pulmonologist include bronchoscopy, incremental exercise testing, and high altitude.<sup>4–6</sup> The use of the phosphodiesterase inhibitors sildenafil and tadalafil for erectile dysfunction has been associated with cases of TGA.<sup>7</sup> The mechanism of action may be secondary to the production of nitric oxide donors, which can precipitate migraine. Alternatively, sexual intercourse may increase sympathetic tone, venous return, and lead to higher spikes of venous pressure during Valsalva maneuvers. TGA has not been reported following the use of these medications for pulmonary hypertension.

TGA is a well-known disorder to neurologists but is less recognized by other physicians, such as pulmonologists, who may be the first to encounter it as a complication of a procedure or medication. TGA, with its heterogeneous set of triggering events, may have a multifactorial etiology, and increasing recognition will only help to enhance its understanding.

## Conflicts of interest

None of the authors have any conflicts of interest to report.

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## Contributions

Dr. M. Robbins was responsible for the identification of the subject, interpretation of the data, drafting of the manuscript, and critical revision. Dr. D. Breidbart was responsible for the identification of the subject, interpretation of the data, and critical revision. Dr. H. Robbins was responsible for the interpretation of the data, drafting of the manuscript, and critical revision.

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